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Pathophysiology and Management of Pain in Children

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Key words

Pain, Pain management

Introduction

Many people avoid seeking dental care because they are afraid of pain that accompanies some dental procedures as a result of tissue trauma and the subsequent release of potent inflammatory mediators which cause pain. Good management of pain and anxiety in dental practice, especially for children, is a corner stone of successful paediatric dentistry and promotes positive patient compliance.

In order to manage the pain the dentist should have clear knowledge of pain neurophysiology, type of pain, causes of pain, mechanism of pain, and the relationship between pain and inflammation. Furthermore, understanding of pain measurement tools and pain control approaches (non-pharmacological and pharmacological) is essential. The role of complementary medicine and itis relationship to the drugs used for controlling pain in dentistry should be clear for the treating dentist. This paper will discuss the different aspects of pain control in children.

Neurophysiology of pain

Pain is a feeling of distress, suffering, or agony caused by stimulation of specialised nerve endings, or an unpleasant sensory and emotional experience associated with actual or potential tissue damage.[1,2] Pain is a protective mechanism for the body, occurring whenever tissues are being damaged so that the individual will react to remove the pain stimulus.[1]

The way in which an individual will react to pain is determined by previous painful experiences in early life.[3] Inadequately-treated pain in childhood may cause a negative impact on the person's response to medical and dental care later in life.[3] Successful dental therapy requires knowledge of the anatomy and physiology of pain pathways, and the materials used to control pain. Such understanding requires a comprehension of the inflammatory mediators, neurotransmitters and relevant nervous system pathways involved in the perception and modification of pain and of the clinical pharmacology of therapeutic agents which control pain, as well as basic knowledge about human psychology.

Classification of pain

Pain can be divided into two broad categories: adaptive and maladaptive.[4] Adaptive pain is the type of pain that determines the survival of the body by protecting it from injury and promotes healing when injury has already occurred.[4] Maladaptive pain, in contrast, is pain presenting as a disease, where pain is an expression of the pathologic operation of the nervous system.[4]

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THIS ISSUE


Nina Vasan

President's Report

The year is rushing by, with the cold winter weather arriving (unless of course, you live in warm Perth!). Coughs, colds and flus are all going around. Everyone in our household has been affected. I think it starts from Sasha now two years old, sharing her dummy with her 'friends' at daycare. I have witnessed, one child dropping their dummy while as fast as hungry vultures, another child picks it up and starts to madly suck on it. It doesn't stop there! The new batch of playdough made every morning with bits of glitter has been sampled by every child and millions of S. aureus multiply happily by the end of the day. I think it is best to drop of the child and quickly run out before you start to see too much!

The future of Paediatric Dentistry

The future of Paediatric Dentistry lies in the hands and minds of the new graduating Paediatric Dentists. In April I attended the AAPD meeting in Sanctuary Cove and it was great to meet many of the postgraduates. There was one day specifically for Postgrads to attend which provided an opportunity for them to discuss issues, problems and generally get to know each other better (even if that means swimming in the hotel pool at 2am!). In the last issue of Synopses many had written about their research projects. They are a neat bunch of people enthusiastic about paediatric dentistry and I'm sure they will continue to move Paediatric Dentistry forward in Australasia.

Reflective Practice

I was reading the latest Dental Protection annual review 2008 (which can be an unsettling read at the best of times!). This issue was titled 'On Reflection'. As dentists most of our early dental knowledge comes from books and journals, rote learning at University. Later, learning comes from clinical experience and reflective practice. Reflective practice is a way of looking back at different situations, analysing why something went well or did not achieve the desired outcome and learning from that experience. The hope is that a practitioner who incorporates reflective learning as part of their regular day to day practice will reduce the risk of adverse events and complaints. This issue looks at reflecting on different aspects of the practice, such as: adverse events (taking out the wrong tooth!); the records and consent; personal reflections; team members; the patients view etc. It is well worth a read for the whole team.

Sudden Infant Death Syndrome

Recently at our practice we received some sad news regarding one of the anaesthetists we work with. His infant son was put to sleep in his cot on Saturday night and the next morning was found dead. What do you say to someone who has lost their child?

So I did some reading into this devastating condition. Sudden infant death syndrome (SIDS) is one of the leading causes of death for children under one. The Australian Bureau of Statistics figures show that there was an average of 210 deaths per year from SIDS. The mortality has decreased over time with more awareness, but SIDS still causes more deaths than traffic injuries, congenital anomalies and cancer combined in the 1-4 years age group.

The cause or causes of SIDS remain largely unknown, although the most likely mechanisms include airway obstruction, re-breathing of expired gases, thermal stress and an 'arousal defect' (reduced ability to respond to hypoxia or hypercapnoea by arousing or waking up). The number of SIDS cases peaks at 8 weeks to 10 weeks of age. It's a time-frame when infants are particularly vulnerable as antibodies crossing the placenta from mother to baby are starting to disappear and babies have yet to produce a significant level of their own antibodies.

In the latest The Lancet Journal Professor Nigel Klein from the Great Ormond Street Hospital for Children in London and his team had found bacteria such as S. aureus and E. coli in nearly half of all babies who died suddenly and without explanation over a decade at a London hospital. Although the presence of bacteria does not indicate a cause, it may help identify a potential risk factor. The bacteria in combination with other co-factors might result in SIDS.

Several factors have been identified that increase an infant's risk for SIDS:

1. Tummy (prone) or side sleeping
2. Sleeping on soft surfaces (waterbed, soft, pillows) or loose bedding
3. Overheating from overdressing, too many blankets or a hot bed room.
4. Smoking in the home or during pregnancy
5. Preterm and low birth weight infants

Some upcoming events to mark on your calendar:

- The NZDA biennial conference is being held in Rotorua, September 10-13. It's nice to see one of the keynote speakers is our very own Dr Kareen Merketichian. I'm sure Kareen will impress the audience and raise the profile of Paediatric Dentistry.
- Prior to the next ADA congress in Perth 2009; ANZSPD and the AAPD will be holding a combined pre-congress day on Thursday, 12 March. The theme being "Early

Childhood Symposium – "Catch them young and treat them well..." This promises to be a unique interactive day with multiple speakers.

- I received a warm invitation from Dr John Rutkauskas, Executive Director for the American Academy of Pediatric Dentistry, he wrote: "I would like to invite you and members of the ANZSPD to the 62nd Annual Session of the American Academy of Pediatric Dentistry scheduled for 21-23 May 2009 in Honolulu Hawaii. It is our strong hope that we will have a significant presence of our colleagues from throughout the Pacific Rim. The programme will be outstanding and it is becoming clear already that we may well have a record attendance from our own membership ranks." I have not been to an American Academy of Paediatric Dental Congress, but I have heard they are impressive and worth making the effort to attend.

Something to think about

"Look at everything as though you were seeing it either for the first or last time. Then your time on earth will be filled with glory."

Betty Smith, Novelist

Keep warm – or come and visit us, our ski season has now officially started!

Nina Vasan

Sedation in Paediatric Dentistry Seminar • 10 October 2008 • Sydney Australia

The Organising Committee, Dr Eduardo Alcaino and Dr Peter Wong, are proud to host the inaugural "Sedation in Paediatric Dentistry" Seminar to be held at the Crowne Plaza, Darling Harbour, Sydney on 10 October 2008.

This seminar aims to provide delegates with:

- A definition of sedation and the forms of sedation available
- Information to dispel the myths and misnomers concerning sedation
- Training from a world renowned Paediatric dental sedationist
- Details on the rules and regulations governing sedation in Australasia
- Education on the forms of sedation you can safely perform in practice
- Current information on morbidity and mortality concerning dental sedation.

Speakers & Themes

The Organising Committee is pleased to confirm that **Prof Stephen Wilson** of the Cincinnati Children's Hospital Medical Center (USA) will be the Keynote speaker at the 2008 event. A/Prof Doug Stewart, Head of the Sedation and Pain Control Unit at Westmead (WCOH), paediatric dentists and other medical specialists will also be sharing their expertise.

Clinical and didactic areas to be covered will include:

- Child development & behaviour management
- Case reports & Adverse reactions
- Guidelines, monitoring, providers of sedation in AUS
- Mortality and Morbidity in Paediatric Dentistry
- Definition of Sedation, Informed Consent, and Assessment of the Paediatric Patient
- Drugs used in Oral Sedation, Relative Analgesia, and new research in paediatric dental sedation

Who Should Attend?

The seminar is expected to attract over 100 participants. If your practice involves the treatment of children, or have patients treated under sedation then you must attend this seminar. This seminar is open to dentists, registrars, and all auxiliaries who have an interest in treating children or who already treat patients under some form of sedation.

For more information and to express your interest:

For more information on speakers and the program or to register your interest please visit the conference website on **www.sydneypaediatricdentistry.com.au/2008seminar** or contact the Conference Secretariat on **spd2008@conexion.com.au**

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Another way of classifying pain is according to the underlying pathophysiologic features. There are two main categories: nociceptive pain and neuropathic pain. Mechanical, thermal or chemical activation of the nociceptive afferent receptors results in nociceptive pain, which can be further classified as somatic or visceral. Somatic nociception is well localised and involves pathologic conditions of skin, muscles, fascia, teeth and bones. For example, the pain associated with cavity preparation or periodontitis involves somatic nociception. In both these situations the nociceptive receptors are sensitised or activated by inflammatory mediators, resulting in transduction of the noxious stimulus into electrical and biochemical signals between neurons. The resultant electrical signal is then conducted to the brain for interpretation as pain sensation.[5] Visceral nociceptive pain is usually poorly localised and involves pathologic conditions in deep visceral tissues such as angina which results from myocardial ischemia. This pain may be referred to the superficial somatic regions and in the case of angina pain may be referred to the jaw, neck, or arm.[5]

Neuropathic pain is characterised by electrical shock which resembles a pin prick feeling on a background of blazing sensation that might be a result of over activity of the nerves in the peripheral nervous system or the central nervous system (CNS).[5] Two examples of this type of pain in the orofacial region are trigeminal neuralgia and post-herpetic neuralgia. Management of orofacial pain of neuropathic origin generally requires more complicated diagnostic testing in clinical practices specialising in treating pain.[5,6]

Further sub-classifications of pain are possible according to different features: duration (acute or chronic, transient, intermittent or persistent), intensity (mild, moderate or severe), quality (sharp, burning or dull), referral (superficial or deep, localised or diffuse).[4,5] Pain can also be divided into fast pain and slow pain. Fast pain (also known as sharp, pricking, acute and electric pain) is felt within a fraction of a second after the stimulus is applied, such as in the case of acute reversible pulpitis or dental hypersensitivity. This type of pain involves the superficial organs and is not felt in most of the deeper tissues of the body.[7] Slow pain (also known as burning, aching, throbbing, nauseous or dull pain) in contrast is usually associated with tissue destruction. Slow pain can lead to prolonged, unbearable suffering such as in the case of acute irreversible pulpitis.[7] Pain might result from different causes such as tissue ischemia, muscle contraction, bacterial toxins effect (dental caries) and direct trauma (cavity preparation).

Mechanisms of pain

Nociceptive pain occurs as a result of articulation of four essential processes; transduction, conduction, transmission and perception. Transduction occurs in the peripheral terminals of nociceptor sensory fibres and involves the conversion of noxious thermal, mechanical or chemical stimuli into electrical activity. The mediators of this process are specific receptor ion channels that are expressed only by the nociceptors.[4] The process of the flow of action potentials from the peripheral terminals along axons to the central terminals of nociceptors in CNS is called conduction. Transmission is the synaptic transfer and modulation of input from one neuron to another.[4]

Pain perception occurs when the noxious stimuli (damaging stimuli that can produce tissue damage) are detected by the terminal endings of two major classes of nociceptive afferent fibres called A delta and C fibres. These fibres are free nerve endings and pain receptors that are distributed throughout the skin, oral mucosa and tooth pulp.[8] The A delta nerve fibres are lightly myelinated and conduct fast sharp pain signals that result from either mechanical or thermal stimuli.[7,8] The C nerve fibres are unmyelinated fibres that transmit slow pain caused by thermal, mechanical and chemical stimuli.[8]

Many factors such as psychological, physiological, emotional and behavioral factors influence the perception of and response to pain. These aspects vary widely among individuals. This might be reflected in the interpretation of the nociceptive signals and determine the intensity of pain. Consequently, the degree of tissue damage observed does not necessarily reflect the intensity of pain felt by the individual.[2]

Pain pathways

The perception of pain by the cerebral cortex occurs via a specialised high-threshold sensory system called the nociceptive system, which mediates the noxious stimuli. This system extends from the periphery through the spinal cord, brain stem and the thalamus to the cerebral cortex where the sensation is perceived.[4] This early warning system protects the body and should be controlled only under specific clinical situations; for example during surgery or medical procedures that cause iatrogenic tissue damage or following trauma. When the protective function of the nociceptive pain system is lost due to a loss of high-threshold sensory neurons, ultimate tissue damage might occur such as in self-induced mutilation. An example of this condition is the mutation of the nerve growth factor tyrosinekinase A receptor in patients with congenital insensitivity to pain. This might reduce the life expectancy of these patients.[4]

There are two separate pathways for transmitting pain signals to the CNS: a fast sharp pain pathway and a slow-chronic pain pathway. When an organ is exposed to a sudden painful stimuli, a double pain sensation felt. The first sensation is a fast, sharp pain that is transmitted to the brain by the A delta fibre pathway, followed by a slow pain that is transmitted by the C nerve fibres pathway. The sharp pain plays an important role in making the person react immediately to remove him or herself from the stimulus. The intensity of slow pain increases over time and causes an intolerable suffering sensation of long continuous pain making the person continue to try to alleviate the origin of the pain.[7,8] There are two pathways for pain transmission from the spinal cord to the pain perception centers in the cerebral cortex. The first pathway is the neospinothalamic tract, which transmits the fast pain, and the second one is the paleospinothalamic tract, which transmits the slow pain.

Pain and inflammation associated with dental procedures

Many dental procedures such as periodontal treatment, endodontic treatment and tooth extraction produce tissue damage and subsequent inflammatory response. This response includes the classical signs of inflammation: pain, oedema, local increased temperature, redness and loss of function. Inflammation is an essential step for healing. When there is an inflammatory reaction in a tissue, there might be an increased sensitivity to the normal stimuli that did not cause

pain prior to the inflammation. This occurs because inflammation and pain are highly articulated processes. Pain occurs due to the action of the central and local inflammatory mediators on the nerve endings. In order to minimise the post operative inflammation and to provide better pain control it is essential to understand the basics of the process of inflammation. This will lead to the provision of the suitable therapeutic measures that minimise the synthesis or release of local inflammatory mediators.[8]

When tissue damage occurs, several inflammatory mediators are released from the damaged cells and from the blood stream. Inflammatory mediators released from the cell membrane include arachidonic acid, prostaglandins, thromboxane A2, leukotrienes (which increase hyperalgesia), phospholipase A2 and histamine. The characteristics of hyperalgesia are spontaneous pain, decreased pain threshold and the perceived pain for any given stimulus magnitude.[5,8] Several electrophysiological changes occur in the peripheral nerve endings and in the CNS. The inflammatory mediators decrease the pain threshold of the peripheral nerve endings to a degree that the normal stimuli that were not causing pain now do so. An example of this hyperalgesia is the throbbing pain of pulpitis, which occurs due to the wave of the arterial pressure following a heartbeat, and the supersensitive nerve endings transmit this sensation as pain.

Another effect of the inflammatory mediators is to stimulate the release of neuropeptides stored in the peripheral nociceptive nerve endings. These neuropeptides are highly concentrated in the dental pulp nerves, promoting the cyclic release of more inflammatory mediators. The combined and constant actions of peripheral inflammatory mediators cause prolonged pain. This extended pain course occurs due to the accumulation of plasma extravasated fluid that results from vasodilatation of local blood vessels and increased tissue permeability. This accumulation of plasma extravasation produces oedema and refreshes the supply of more inflammatory mediators that in turn increase the accumulation of plasma extravasations.[8]

Measurement of pain

The measurement of pain remains a challenge for medical and dental professionals because there is no accurate objective tool such as certain clinical or physiological signs that can quantify the amount of pain an individual is feeling. The early investigations more than a century ago suggested that pain consisted of both cognitive and affective components making it difficult to be measured.9 The medical professional might assess the patient's pain, but this may lead to underestimation of the intensity of pain.[2] According to the American National Institutes for Health (NIH), patient self-reporting is the 'most reliable indicator of the existence and intensity of pain'.[2] An ideal subjective pain measurement tool should identify the presence or absence of pain; pattern of pain progression with time and treatment, quality, impact and personal meaning of the pain. In order to validate a pain measurement tool it should be applied equally to individuals despite psychological, emotional and cultural background differences.[2]

A number of valid pain measurement tools have been developed, which are also called unidimensional pain scales.[2] According to a review of the literature from 1992-2002 there are five popular pain scales: the visual analogue scale (VAS), verbal rating scale (VRS), numeric rating scale (NRS), Mc Gill pain questionnaire (MPQ), the brief pain inventory (BPI) and the 'Wong-Baker faces scale'.[10] Children over 4-5 years of age can self-report of pain by VAS using faces, while older children can use an adult VAS rating of 1-10.[10, 11]

In certain circumstances subjective pain reporting is not possible such as in the absence of sufficient spoken skills in neonates and children younger than three years who cannot distinguish between pressure and pain, in the cases of intellectually disabled patients, and during the postoperative period when the patient is still under the influence of anaesthetics. In such cases, the assessment of the medical professional can substitute the pain measurement scale but cannot give the same accuracy. This assessment is based on physiologic and behavioral responses such as blood pressure, crying movements, agitation and verbal expression/body language.[2,12] Parents are the best reference to estimate the pain response of a child. Other substandard methods that been reported to objectively measure pain intensity are the mechanical methods such as squeezing a calibrated tool to express the amount of pain.[2]

Control of pain

There are many factors affecting the perception of pain and the reaction to it. These factors are a complex combination of physiological, pathological, emotional, psychological, cognitive, environmental and social factors.[12] There are two approaches to deal with pain in paediatric dentistry setting; these are non-pharmacological and pharmacological approaches.

Non-pharmacological approach to pain control

The non-pharmacological approach depends on the good understanding of the basis of the distress associated with a child presenting to the dental surgery complaining of pain. The term distress describes the mixture of fear, anxiety and pain.[13] There are several factors that determine the degree of the distress the child might have towards dental pain and treatment. These factors might be individual, parental, situational or a combination of all these factors.

The individual factors depend mainly on the age and cognitive ability of the child, as this determines the perception, understanding, memory and reporting of pain. The younger the age and the less the cognitive ability the more the degree of distress the child may have.[13] There is a strong positive association between the parental anxiety and the degree of the child's distress. The treatment venue setting and the attitude of the professional staff towards young patients and their parents, and the so-called situational factors also affect the degree of the distress of the child. For example, the setting of instruments such as needles within the child's sight and non-friendly staff can increase the degree of the distress of the patient. There are many psychological approaches to decrease the anxiety levels such as positive reinforcement, guided imagery distraction, graded exposure, relaxation by breathing control. These psychological approaches are also called cognitive behavior therapy (CBT) and systematic desensitisation.[13]

Distress can be prevented by proper preparation of the child by the parents prior to the dental appointment to consider it a positive experience. The

parents should know in advance what to expect in the dental surgery and the type of treatment the child will receive by providing them with sufficient information in advance. At the appointment, listening to the child is very important in assessing the degree of cognitive ability and the effects of any previous or dental experience. During the treatment the child should be given some control such as raising a hand if he/she wants the dentist to stop or plan in advance if the child wants to make noise while having an injection for example. In cases of anxious children the presence of many people around the child might increase the degree of the distress so having few individuals present is a wise idea.[13]

Pharmacological approach to pain control

There are four pharmacological approaches to control pain: analgesia, local anesthesia, sedation and general anesthesia. Each of these methods has its specific indications and contra indications according to the pain cause and the proposed treatment. The main purpose of the pharmacological pain control is to disturb the nociceptive processes at any of the three stages of the process: initiation of impulses, propagation of those impulses and perception of painful stimuli by using different types of drugs and methods of administration.[5]

Analgesia refers to the process of relief of pain without loss of consciousness by administration of pain killers or analgesics such as non-opioids, opioids or a combination of the two. Non-opioid analgesics include paracetamol and non-steroidal anti-inflammatory drugs (NSAIDs). Opioid analgesics are narcotics such as morphine, pethidine and codeine phosphate. Administration of analgesics preoperatively may decrease postoperative pain as it minimises the synthesis of prostaglandins that are released as an immediate reaction to the surgical procedure.[5]

Paracetamol has an anti-pyretic effect but is not anti-inflammatory and has rare side effects including rash and blood dyscrasias.[14] Paracetamol might be contraindicated in certain cases such as in hepatic and renal impairment. The recommended dose is 15mg/kg up to 90mg as maximum dose per day.[11] The dose can be repeated at 4-6 hour intervals when necessary.

The non steroidal anti-inflammatory drugs (NSAIDs) are the most widely used pain medications for pain associated with dental procedures and problems. The main action of NSAIDs is to minimise or prevent the synthesis and release of inflammatory mediators such as bradykinin. These inflammatory mediators sensitise the nociceptive receptors at the site of injury at which the initiation of nociceptive impulses occurs in the periphery. There is a distinct specified group of NSAIDs called COX-2 inhibitors which selectively block the enzyme cox-2 (cyclooxygenase-2). Blocking this enzyme impedes the production of the prostaglandins. It is advisable to administer NSAIDs preoperatively to delay the onset of postoperative dental pain and decreases its severity. This will lead to lower dose of analgesic required postoperatively.[5]

The main side effects of NSAIDs are dyspepsia and nausea. There are some rare serious side effects such as allergic reactions, bronchoconstriction in asthmatics and prior gastrointestinal perforations, ulcerations or serious bleeding reactions. The negative reaction to one of the NSAIDs does not necessarily mean that the patient would react to all types of NSAIDs. There are some superior advantages of COX-2 inhibitors over other NSAIDs, such as lower incidence of serious upper gastrointestinal toxicity (symptomatic ulcers, bleeding, and perforation) decreased nephrotoxicity and a lack of effect on platelet aggregation.[5]

There are different preparations of NSAIDs as tablets, capsules or liquid formulation. The appropriate form should be prescribed according to the patient's age and physical ability. The liquid formulation is preferable for children. The most popular type of the NSAID is ibuprofen. The ideal dose of ibuprofen for a child is 5-10 mg/kg every 8 hours.[11]

Opioids and compound analgesia

The action of opioid analgesics is centrally in certain loci in the CNS. Opioid analgesics decrease the perception of pain by affecting certain receptors in the spinal cord, rostroventral medulla and periaqueductal gray matter. For severe pain or when non-opioids are not effective, opioid analgesics can be added in combination as compound analgesia. The main advantage of compound analgesia is the lower doses of medications required for pain control.[5] Compound analgesia means either a dose of opioid and non-opioid taken at the same time or alternating the opioid with non-opioid in overlapped doses, to assure stable plasma levels of the analgesics for the required period of time (for example, for 48 hours).[5]

Local anesthesia

Local anesthetics (LA) are used routinely in dentistry. The essential action of LA is preventing the nociceptive stimuli from reaching the brain or the spinal cord, resulting in blocking of the propagation of nerve impulses from the injured site to the higher centers in the cerebral cortex.

The available LAs can be short acting or long acting and LAs can be administered topically, locally or regionally. For dental procedures in children the most commonly used LA is short acting (e.g. Xylocaine™) for topical and local applications. The long acting LA (e.g. bupivacaine) is usually used for adults rather than children under the age of ten. The application of LA can have a favorable effect in reducing the onset and intensity of postoperative pain.[5]

Relative analgesia

Relative analgesia (RA) refers to the use of a sedative agent either by inhalation, intravenous, oral route of administration. The most widely used sedative by inhalation is nitrous oxide (N_2O). This agent is considered safe to be used in the outpatient setting with the need for simple monitoring devices. Recovery is almost immediate after the cessation of the N_2O. The use of N_2O requires special training and the use of a pulsoximeter to monitor the oxygen saturation in the patient's blood during the procedure is a vital requirement. Careful patient selection for N_2O sedation is mandatory because it will not be effective in very young children or uncooperative patients. The use of N_2O can help patients to overcome their anxiety towards dental procedures.[11] Other forms of RA (e.g. intravenous, rectal, intramuscular or oral sedatives) might need to be used in a hospital setting with an anesthetist. In all forms of RA a resuscitation kit should be available on hand for any unexpected complications.

General anesthesia

Performing dental treatment under general anesthesia is considered the last option for treating an uncooperative child after all the conventional measures of management have failed. This decision should be based on careful weighing-up the risk of and benefits of the procedure. General anesthesia can be a good step of behavior management by eliminating the sources of dental pain and this may make the child cope better subsequently with the dental setting.[11]

Complementary and Alternative Medicine

Complementary and Alternative Medicine (CAM) refers to a variety of therapeutic or preventive health care practices that do not follow conventional medical measures and may not have an evidence-based, scientific explanation for their effectiveness. The CAM include homeopathy, naturopathy, chiropractic, and herbal medicine.[16]

There are few published reports that describe and document various herbal medications used for treatment of dental problems and their implications in conventional dental management.[17] The reported dental implications vary from drug interaction including allergic reactions, hepatotoxicity, mutgenicity, bleeding episodes and cardiac abnormalities and even death.[17] Some of the reported drug interactions include adverse effects when using herbs in association with traditional medications such as cardiovascular (CV) medications, NSAIDs and antidepressants. Certain herbs can decrease the effect of CV medications or exacerbate the effect, for example, an increase in the anti-platelet effect of certain CV medications. The documented herbs that can produce these negative interactions include ginseng, gingko biloba and St. John's wort.[18]

Use of ginseng, gingko biloba and garlic in conjunction with NSAIDs can enhance the damage to the gastro-intestinal tract and the anti-platelet effect increasing the risk of bleeding. Gingko biloba and St. John's wort can increase the sedative effect of the antidepressants when used in conjunction with each other.[18] It is recommended that the dentists include an identified section in the dental records documenting the use of herbal remedies including types and names. Dentists should be knowledgeable and fully alert to the adverse effects that can occur when using certain herbs.[17, 18]

Humour and pain

The effect of humour in decreasing pain intensity has been studied and humour may have a favorable effect in controlling pain.[19] An association has been reported between laughter and sense of humour with healing acceleration and boosting of the immune system function.[19] This is thought to be due to prompting the production of endorphins, and the reduction of the production of the stress hormones such as cortisol and adrenaline, by laughter.[19]

A 1998 study in California, USA, showed that children and teenagers with chronic illnesses who viewed comedy movies had improvement of their immunity.[19] Other research confirms that a positive attitude plays an important role in recovery during illness.[19] Another American study on healthy children who were connected to monitoring devices and then asked to do something harmless but painful (such as submerge a hand in cold water at 10°C, keeping it there as long as possible). Those viewing comedy videos during the procedure were able to hold their hands in water 40 per cent longer than children who did not see a video.[19] Other findings indicated that children and teens were significantly better able to withstand painful procedures while they were watching and laughing at humorous programmes. An endorphin response can be triggered by making patients smile and laugh.[19]

Summary

Pain control in children involves several factors. A good understanding of pain control requires familiarity with the basic neurophysiology of pain, child psychology and the pharmacology of analgesics and local anesthetics. Today herbal remedies should be acknowledged and dentists should ask about its use prior to prescribing medications or using LA. Humour in dental practice is can be a valuable adjunct to medical approaches to pain management and particularly in children.

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Soft Molar Study

A study looking into the prevalence of soft molar teeth in New Zealand children is currently underway in Wainuiomata, Wellington. Dr Erin Mahoney, Paediatric Dentist and Research Assistant David Morrison have been visiting the primary schools in the region and examining children – in the classroom. They are now halfway through their examination process. The purpose of this article is to outline the study and provide a few anecdotes from their school visits.

Dr Erin Mahoney

David Morrison

Background

Developmental defects of enamel (DD) are an increasing concern in children1. DD are often caused by a systemic insult during tooth development, the most commonly affected teeth being the first permanent molar. These are seen either as yellow-brown demarcated opacities (enamel hypomineralisation) or as a total lack of enamel (enamel hypoplasia). Defects in molar teeth significantly increase dental treatment need in young, often nervous, children and frequently result in extraction. This has a major impact on the overall dental health and wellbeing of these children. Affected teeth are also considered to be more susceptible to dental caries, although the reason for this is presently unknown.[2,3]

Aims

Dr Erin Mahoney from Hutt Valley DHB and I, David Morrison from the Dental Research Group, are conducting a two-part HRC funded study looking into several key issues regarding DD. The first part is a prevalence study that aims to determine the frequency of DD in New Zealand children and assess any differences between Maori, Pacific Island and Pakeha. The second part is a laboratory study that aims to determine if i) hypomineralised enamel is inherently more susceptible to dental caries than healthy enamel and quantify the differences, and ii) if fluoride and calcium phosphate-monofluorophosphate urea (CPMU) mineralising solution can reduce the development of caries or the severity of an already present carious lesion in hypomineralised enamel.

Presently there is very limited information about the prevalence of DD in New Zealand children, although re-analysis (unpublished) of a previous NZ study in Southland found that approximately 6% of children had hypoplastic or hypomineralised defects in their first permanent molar teeth.[4]

Soft Molar Teeth in Wainuiomata Children, the study so far…

The prevalence study is taking place in the Wainuiomata basin situated in the Lower Hutt region of Wellington. This region was chosen because of its comparatively large Maori and Pacific Island population; approximately 30-40% of the population is Maori, 10-15% Pacific Island and the remaining majority being Pakeha. There are roughly 800 students in our target age group in the region. All 7-10 year old children in the region were invited to take part in the study. This was done through school newsletters, an information evening and information sheet and parental consent form sent home with the children.

Prior to the project getting funding Dr Mahoney had been in discussion with the school Principles' about the study, gaining consent and working out the logistics of our visits. Erin and I also met with the local Iwi at the Waiwhetu Marae. All the parties involved were very supportive of the study as dental problems are common in the Wainuiomata region. Contact with these parties is maintained throughout the study via study updates, thank you letters and later, a summary of our findings. These liaisons proved invaluable as the other parties all offered good advice; locations for hosting an information evening, contacts at local paper and radio stations etc. The local papers and the radio station also enthusiastically ran articles about the study. The effectiveness of this press is hard to measure, our hope was that it would raise awareness about teeth, our study and act as reminder to parents/ caregivers to read and return the consent forms.

Without face-to-face contact with parents' gaining consent was difficult. A reminder letter and additional consent forms were sent home a week prior to our visit. These proved crucial in gaining consent and raised returns from 45% to 63%. We gave our information sheets and consent forms to the school receptionists' who did a wonderful job of circulating to, and collecting from, the classrooms.

Data collection has to date involved Dr Mahoney and I visiting the classroom and, after a quick chat about oral hygiene, we examine the children's teeth. We set up anywhere we can, usually at the front of the classroom. All we need is a table and chair although at some point during our school visit we require a sink and water to make up a cold sterilisation bath used to clean the equipment. Child assent forms are handed out on our arrival. Getting theses signed was not difficult. In one classroom a teacher continued to read to the class while we carried out our examinations in hushed tones. One-by-one those students with parental consent are called and examined by Dr Mahoney. We record any enamel opacities, total absence of enamel and decayed, missing or filled teeth. If during the examinations Dr Mahoney notices any problems such as supernumerary teeth, vulnerable hypoplastic molars etc. she has informed the local school dental therapist. Dr Mahoney was calibrated prior to starting the study with another Paediatric Dentist using photographs and the examination of 10 patients with and without molar hypo-mineralisation. We are to return to a school shortly where Dr Mahoney will re-examine a classroom, n=15 students, to assess intra-examiner reliability.

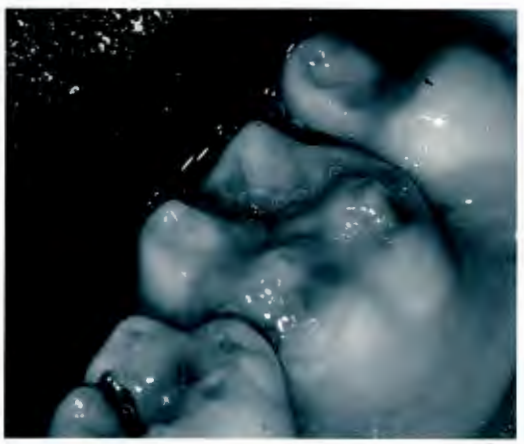
Children anxious about our exam have been few and far between. So far, only one has declined. It seems once the first child has been seen to 'survive' the exam everyone else is happy to line up. In fact, our problem is the opposite, children without parental consent wanting to have the exam, "hey Miss, you forgot about me!?" Rebecca Schipper from Colgate has been very supportive of the study and, much to the children's delight, Colgate has kindly donated toothbrushes and paste

for each child. There was often a collective "Oooo" when this was announced. These were given to all the students regardless of participation. They were genuinely excited about receiving the brushes and paste – so much so that we had to start giving the paste to the teacher to prevent in-class teeth-cleaning mayhem ensuing!

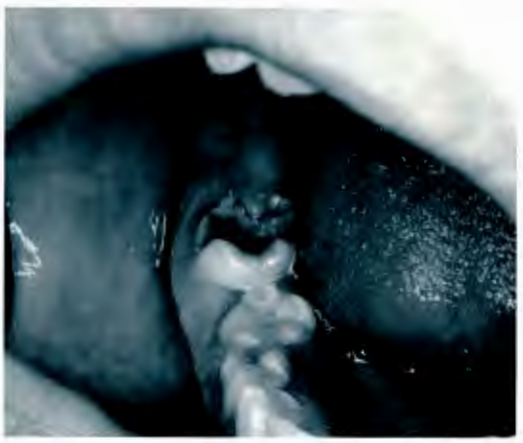
The school examinations so far have been painless and our classroom visits of minimal disruption. Both Erin and I are constantly surprised at how helpful and compliant the children and teachers are. On average each child took a minute to examine, a classroom was done in 25-30 minutes and most schools were completed by lunchtime.

Our method of gathering data has proved to be a success. It does require some groundwork; building relationships with the schools, publicising the study locally and circulating study information and consent forms. Once this is done the actual visits are a fun, fast and efficient way to gather data. It is mutually beneficial – we get our data and the children get some dental education and an additional oral examination. Soon we will be moving onto the data analysis, but before this, Erin and I look forward to visiting the final three schools.

Dr Erin Mahoney with Room 4, Arakura Primary School, Wainuiomata



Enamel Hypomineralisation



Enamel Hypoplasia

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CANADIAN DENTAL ASSOCIATION
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Clinical Issues – Bisphenol A

Alistair Devlin

There has been some discussion about bisphenol A toxicity in recent times. I received this amongst other documents from our Canadian friend and colleague Prof David Kenny in Toronto. Dave describes the issue as having erupted in Canada in recent months. I hope you find this interesting.... Alistair Devlin

Background

Safety concerns about bisphenol A (BPA) are based on research performed on animals that suggest BPA might affect reproduction and development in humans by acting like estrogen.

Health Canada conducted a risk assessment on BPA and announced its results on April 18. The focus of the report's recommendations was on polycarbonate baby bottles and infant formula containers, not dental materials.

The report concluded that BPA exposure from dental sealants and composites is extremely small in comparison to other sources and that no further regulations on BPA derived from these materials is required.

Media coverage leading up to the report confused the issue by listing 'dental sealants' along with other household products that may release BPA. Despite the report's findings, negative public opinion may outweigh science and prompt questions or concerns from patients.

Main messages

Dental sealants are an excellent method of protecting against cavities and the need for future restorations – particularly in the young patient.

Based on the current evidence, there is no cause for concern regarding BPA released from dental composites or sealants. Exposure from these materials is significantly lower than from other sources of exposure.

The amount of BPA released from dental sealants or composites is extremely small and limited to a small number of products. The minimal exposure generally occurs within the first few hours after placement and then quickly reduces to virtually nothing. Once the material is placed and hardened, it becomes very stable.

Health Canada's provisional standard on BPA levels in humans is 25 micrograms daily per kilo of body weight. No restrictions are planned for dental composites or sealants as potential exposures of BPA from these materials fall well within Health Canada's safety margins.

Supporting Arguments

While the term 'bisphenol' is found in the name of a large number of chemical compounds, the only compound reviewed by Health Canada was bisphenol A (BPA). Other bisphenols in general, and bis-GMA in particular, are not a concern.

Some of the most effective, proven materials for dental sealants are resin-based. BPA is not used as an ingredient in the manufacture of resin-based dental sealants. Dental sealants may contain monomers that are derived from BPA, such as bis-GMA and bis-DMA, but BPA itself is not an ingredient in dental sealants.

Some research has found that bis-DMA (Bisphenol A Dimethacrylate, also known by the acronym 'BAD') is hydrolyzed to BPA. (Schmalz et al, 1999). Other derivatives of BPA, such as bis-GMA and bisHPPP, show no capacity to be hydrolyzed back into BPA under normal conditions in the mouth (Santerre, 2007).

Recent studies have indicated that the exact resin formulation of the sealant may affect the potential for release of BPA. "No BPA-release is expected under physiologic conditions from fissure sealants based on bis-GMA if pure base monomers are used." (Schmalz et al 1999).

Other research has detected low levels of BPA in the saliva of individuals treated with certain dental sealants immediately following its application. (Joskow et al 2006, Sasaki et al 2005). However, no studies to date have detected BPA in the blood stream of individuals.

BPA found in saliva, potentially derived from dental sealants, could come from three sources.

1 Residual Monomer: within minutes of the material being placed in the mouth, unreacted BPA-derived monomer starts to diffuse out of the material.
2 Hydrolysis: hydrolysis of polymerised BPA-derived monomers is catalyzed by salivary enzymes that can break down hydrolytically sensitive ester bonds at the temperature and pH of the mouth.
3 Abrasion: abrasion produces small particles of polymers that are more accessible to the hydrolysis reaction.

All of these processes begin immediately, with hydrolysis and abrasion taking place over a longer period of time. (Santerre, 2007)

Health Canada is currently preparing a database that will identify materials that could potentially release BPA. In the meantime, dentists might find it useful to consult the Material Safety Data Sheets (MSDS) of dental sealants to identify the chemical composition of materials in use.

Clinical Practice Advice

To reduce the potential of any BPA being released from dental sealants, dentists should fully cure the sealants or composites according to manufacturer's instructions. In addition, dentists can treat the surface layer by performing one of the following three procedures:

1 use a mild abrasive, such as pumice, either on a cotton applicator or in a prophy cup;
2 have older children and adolescents gargle with tepid water for 30 seconds; or
3 wash the sealant surface for 30 seconds with an air-water syringe while suctioning fluids and debris from a child's mouth. (Azarpazhooh and Main, 2008).

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Colgate® Corner

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Colgate Bright Smiles, Bright Futures

Welcome Territory Managers


Janne Thomas NSW


Sophie Hughes NSW


Natasha Dosatal VIC


Melissa Wicker VIC


Angela Tascone WA


Lisa Grant NZ

Six new Territory Managers have joined the Colgate team over the past few months (pictured above). All of our new Territory Managers have had experience in dental practices or the dental industry and will be making every effort to meet as many of you as possible over the coming month.

Colgate Chair of Rural Remote and Indigenous Oral Health

We are delighted to announce the appointment of Professor Ratilal Lalloo to the Colgate Chair of Rural, Remote and Indigenous Oral Health at Griffith University, Gold Coast.

Professor Lalloo is a distinguished South African specialist in public oral health, with his dental degree and a Masters of Community Dentistry from the University of The Western Cape; an honours degree in Epidemiology from the University of Cape Town; and a PhD from University College London. He has extensive research experience in population health, in risk factor analysis and in prevention of maxillofacial injury and of dental diseases.

Professor Lalloo has invested a significant amount of time gaining experience amongst the varied communities of South Africa. This experience will equip him well for the challenges of the diverse cultures and needs of rural, remote and Indigenous Australia.

Professor Lalloo will join the staff at Griffith University later this year and take responsibility for the School's Rural Placement Programme and will launch a collaborative research programme in Rural, Remote and Indigenous Oral Health.

Professor Newell Johnson, Dean and Head of the School of Dentistry at Griffith University said "We look forward to welcoming Professor Lalloo to Griffith later this year and the synergisms with Colgate in the promotion of oral [and general] health to a wide community.

NZDA-Colgate Oral Health Educator, Auckland


Deepa Krishnan

Deepa Krishnan has a background in Oral Health and Public Health, preparing her perfectly for this new role in Oral Health Promotion. The first Early Childhood Oral Health Promotion Forum held in late April provided an opportunity for representatives from Dental Health Boards, Public Health Services, Medical Centres, the Ministry of Health, NZ Dental Association and Colgate to share resources and identify opportunities to work as a team more effectively.

Another of Deepa's roles will be to manage the NZ allocation of Colgate toothbrushes and toothpaste provided through the Global Child Dental Health Taskforce. Four DHBs (Northland, Otara, Bay of Plenty, Wanganui and Lakes) with particularly high levels of childhood dental caries, were identified by the Ministry of Health and invited to submit oral health promotion plans. Deepa is working with the DHBs to ensure evaluation criteria are included and the projects should all be underway later this year.

Colgate Territory Managers are here to assist you with the products you need in your surgeries

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Coming events

10 October 2008
Sedation in Paediatric Dentistry
Crowne Plaza Darling Harbour, Sydney

21-25 May 2009
62nd AAPD Annual Session
Honolulu, Hawai

16-20 June 2009
22nd IAPD International Congress
International Congress Centre
Munich, Germany

28-31 March 2010
16th Biennial Congress of ANZSPD
Queenstown, New Zealand

27-31 May 2010
63rd AAPD Annual Session
Chicago, Ill

29-31 October 2008
Paediatric Society of New Zealand
60th Annual Scientific Meeting
Paihia, Bay of Islands

2-3 November 2008
African Regional Paedodontic Congress
Parktown, Johannesburg

Austalian and New Zealand Society of Paediatric Dentistry

www.anzspd.org.au

Federal President Dr Nina Vasan
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Federal Secretary Manager Dr Alistair Devlin
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All text for inclusion in Synopses must be submitted to the editor on floppy disk, zip disk, CD, or by email. Both PC and Mac formats are accepted. Media will not be returned. Address email to dorothy.boyd@phsouth.co.nz. Please enclose your contact details and email address with all submissions.

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